



# Asthma, atopy, and lung function among racially diverse, poor inner-urban Minneapolis schoolchildren

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## Abstract

As part of an assessment of schoolchildren's environmental exposures and health, a probability sample of 136 children from diverse racial/ethnic backgrounds was drawn from grades 2–5 of two inner-urban Minneapolis schools (Whittier, Lyndale). Questionnaires were administered to a parent/guardian; blood samples for IgE and lung function tests were obtained. Overall adjusted rates for lifetime asthma (15.4%; 95%CI 9.3–21.5%), asthma in the last 12 months (13.6%; 7.8–19.4%), and current asthma medication use (10.5%; 5.3–15.7%) were higher than reported US national rates. Adjusted rates for lifetime physician-diagnosed asthma differed significantly among racial/ethnic groups ( $P < 0.01$ ): African-Americans (25.9%), White/Others (25.8%), Hispanics (9.3%), Somalis (1.8%), Asians (0%). Corresponding rates for atopy (total IgE  $> 100$  IU/mL or an allergen-specific IgE  $> 0.35$  IU/mL) were: African-Americans (66.6%), White/Others (100%), Hispanics (77.2%), Somalis (78.1%), Asians (81.8%). Lung function (FEV<sub>1</sub>, FVC) was analyzed by linear regression using log-transformed data: significant race-specific differences in lung function were found relative to White/Others ( $P < 0.001$  for each racial/ethnic group): African-Americans (FEV<sub>1</sub> –16.5%, FVC –16.9%), Somalis (–22.7%, –26.8%), Hispanics (–12.2%, –11.4%) and Asians (–11.1%, –12.4%). Females had significantly lower FEV<sub>1</sub> (–8.8%) and FVC (–11.0%) than males. An unexplained, significant difference in children's lung function was found between the two schools. A history of physician-diagnosed asthma was not associated with decreased lung function. Factors other than poverty, inner-urban living, and IgE levels (atopy) need to be considered in the development of childhood asthma.

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## 1. Introduction

Asthma rates have been increasing in the United States for at least 30 years. This national trend has much of its origins in childhood, with the prevalence of asthma in pre-school children more than doubling in the last 20 years (Mannino et al., 1998, 2002). As a result, asthma presents a major public health problem, with roughly 17 million Americans, including about 5 million children, suffering

from the disease. The trend in the United States is part of an epidemic of childhood asthma that has been documented throughout the industrialized world and is emerging in many developing countries (Beasley et al., 1997; ISAAC, 1998). At the same time, hospital admissions and mortality from asthma have risen (Anderson, 1989; Anderson et al., 1983; Lozano et al., 1999; Newacheck and Halfon, 2000; Pearce et al., 1993; Rasmussen et al., 2002).

The reasons for this asthma epidemic are unclear, although several causes have been proposed, including lifestyle and behavioral factors, childhood respiratory infections, and environmental exposures to allergens,

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airborne particles, noxious gases, and other toxic agents (Kheradman et al., 2002; Leikauf, 2002; Redd, 2002). Higher rates of asthma occur among children living in inner-urban areas of the United States, especially among poor families and certain racial or ethnic groups. It has been suggested that poverty may be more important than race in determining asthma prevalence and the related use of health services (Halfon and Newacheck, 1993; Wissow et al., 1988), although poverty also could be a surrogate for other risk factors.

Racial and ethnic differences in asthma rates in the United States have been well documented, with unusually high rates observed in African-American and Hispanic children (Dey and Bloom, 2005). Asthma rates vary also within racial/ethnic groups with respect to immigrant status and acculturation (Gold & Acevedo-Garcia, 2005). Rates of asthma among Mexican-born Mexican-American children are lower than American-born Mexican-American children (Eldeirawi et al., 2005), lower than Americans of other Hispanic ethnicities, and lower than African-Americans (Dey and Bloom, 2005). These observations are not confined to the US. Studies from Australia, Sweden, the United Kingdom and Israel have also described the importance influences of immigration and acculturation on the rates of childhood asthma. For example, children from Southeast Asian countries who emigrate to Australia arrive with lower rates of allergies and asthma than resident children, which then increase to the level of resident children over a period of 10–15 years (Leung, 1994; Leung et al., 1994). Overall, these findings suggest that environmental factors are responsible for much of the increase in asthma associated with living in an industrialized society (Gold and Acevedo-Garcia, 2005).

Levels of ambient air pollution, with the exception of ozone, have been decreasing in industrialized countries at the same time overall asthma rates have been increasing, suggesting that ambient air contaminants have not been major contributors to the emerging epidemic of asthma in the developed world (Bascom et al., 1996; EPA, 2001; Koenig, 1999). Attention has turned therefore to indoor environments, particularly the home, where exposures to allergens (e.g., house dust, cockroaches, pet dander), emissions from combustion sources, and toxic chemicals may be relevant. Children also spend substantial amounts of time indoors at school, but exposures in schools have rarely been characterized systematically and the contribution of school environments to childhood asthma is unknown.

The School Health Initiative: Environment, Learning and Disease (SHIELD) project examined the environmental exposures of poor, inner-urban children aged 7–12 years in their homes, schools, and ambient air, as well as biomarkers of exposures in blood and urine, and related these to health and learning outcomes. This paper reports data on respiratory symptoms, asthma, atopy, and lung function among these children, with particular reference to racial or ethnic differences. Details of the study design, study population, subject recruitment and participation

have been published (Sexton et al., 2000, 2003), as well as findings for exposures to volatile organic compounds and tobacco-specific metabolites in the urine of these children (Adgate et al., 2004; Hecht et al., 2001; Sexton et al., 2005).

## 2. Methods

The SHIELD study used a probability sample of elementary schoolchildren (grades 2–5) drawn from two adjacent, low-income, racially-mixed, inner-urban neighborhoods in South Minneapolis, Minnesota, USA. The children attended either a school of recent construction, using design features, materials, and maintenance methods that emphasized a healthy indoor environment (Whittier), or a school of traditional design and maintenance that was built in the 1970s (Lyndale) (Sexton et al., 2000, 2003). The sample sizes were determined primarily for the purposes of measuring environmental contaminants and biomarkers of exposures among the children. A secondary goal was to compare the respiratory health of the children in each school.

The study protocol was approved by Human Subjects' Research Committees at the University of Minnesota, the Centers for Disease Control and Prevention, and the Minneapolis Public School District. Informed consent was obtained from each child's parent or guardian, and the assent of each child was obtained immediately before any test was performed.

Trained interviewers administered a questionnaire to each child's parent or guardian in the home. The American Thoracic Society's (ATS) standardized respiratory questionnaire for children was used to determine children's symptoms of cough, phlegm, wheezing, breathlessness, asthma, common allergies, and other respiratory illnesses (Ferris, 1978). Additional questions relating to socioeconomic status and hazardous exposures in the home were included. Local community members of the same race or ethnicity were employed to translate the questionnaires into Spanish, Somali, or Cambodian. Accuracy of the translation was confirmed by independent back-translation into English of each questionnaire. Where English was not spoken in the home, bilingual education assistants and local community members were trained to administer the questionnaires and answer questions from the parents. The lay interviewers were instructed specifically about the confidentiality of any information they helped to collect and about the importance of asking the questions in an unbiased manner.

Lung function tests were administered by school nurses who had completed a NIOSH-certified training program in spirometry testing. Lung function was measured with rolling-seal Survey Spirometers (Warren E. Collins, MA) connected to analog-to-digital transducers and computerized recording systems that stored volume-time data. The spirometers were calibrated with a standard 3-L syringe at the beginning and end of each testing session. Standing height was measured and a brief questionnaire administered

to determine if the child had a current respiratory illness or was receiving medications. Children with symptoms of a respiratory infection were asked to return for testing after recovering from their illness. Forced vital capacity maneuvers were obtained with the child seated; a minimum of three acceptable flow–volume curves were recorded for each subject in accordance with ATS recommendations (ATS, 1987).

Results from at least three technically satisfactory flow–volume curves were stored electronically and subsequently merged with files that included the questionnaire responses, blood and urine results for each child. The largest values for forced expiratory volume in one-second (FEV<sub>1</sub>) and forced vital capacity (FVC) were used in the statistical analyses. The lung function results were compared with predicted values obtained from gender-specific equations for White children (Knudson et al., 1983).

About 30 mL of blood from each child was needed to perform all of the blood tests, but in the younger and smaller children (especially Asians) we often obtained less than 30 mL. Tests for biomarkers of exposure, used by other components of the SHIELD study, were given priority over IgE measurements when an incomplete blood sample was obtained, and thus IgE levels were measured in only 97 (71%) of the children reported here. Total serum IgE, and specific IgE levels to two mite antigens (*D. pteronyssinus*, *D. farinae*), cockroach, cat dander, and ragweed were determined by enzyme immunoassay methods using commercially available analytical kits (Jackola et al., 2002; Patterson et al., 1994).

Statistical analyses were conducted with the SAS<sup>®</sup> software (Statistical Analysis Systems Inc., NC). Categorical data were cross-classified using SAS, and relative frequencies, variances, and standard errors were adjusted using appropriately normalized sampling and response probabilities as weights for individual observations (Horvitz and Thompson, 1952); associations and heterogeneity were tested with Chi-square tests.

Linear regression models for FEV<sub>1</sub> and FVC included continuous variables for height (cm), age (years), and Body Mass Index [BMI = weight (kg)/height<sup>2</sup> (m)], and indicator variables (1,0) for gender (Females = 1), school attended (Whittier = 1), physician-diagnosed asthma, and African-American, Somali, Hispanic, or Asian groups. Two models of lung function were assessed: (i) using raw values for lung function, age, height and BMI; and (ii) using natural logarithms of the same variables. The use of natural logarithms was based on arguments presented by Schwartz and coworkers (1988) for a large analysis of lung function contained in the NHANES II data set. Logarithmic transformation addresses the heteroscedascity present in children's lung function measurements, whereby the variance of the data increases with age and height.

An example of a log-transformed relationship is the following:  $\text{Ln}(\text{FEV}_1) = a + b \cdot \text{Ln}(\text{Age}) + c \cdot \text{Ln}(\text{Height}) + d \cdot \text{Ln}(\text{BMI}) + f \cdot \text{Female} + r_1 \cdot \text{Race}_1 + r_2 \cdot \text{Race}_2 + \dots$ . This can be back-transformed to a linear scale for the outcome

and independent variables:  $\text{FEV}_1 = \exp(a) \times (\text{Age})^b \times (\text{Height})^c \times (\text{BMI})^d \times \exp(f \cdot \text{Female}) \times \exp(r_i \cdot \text{Race}_i)$ , where  $\exp$  is the exponential constant ( $e$ ). The categorical variables for Female and Race take values of one or zero, and it follows that  $e^f$  and  $e^f$  describe the fraction of lung function, on a linear scale, that can be explained by female gender or a particular racial/ethnic group.

### 3. Results

A total population of 558 children were enrolled in grades 2–5 at Whittier ( $N = 269$ ) and Lyndale ( $N = 289$ ) schools. To ensure adequate representation of smaller subsets of the population, the sampling was stratified by school, gender, grade (2,3,4,5), and whether English was the primary language in the home; the number sampled from each of the 32 different cells was equal (Sexton et al., 2002, 2003). The combined cell-specific probability of being sampled and responding was used to weight individual responses in the analysis to adjust results to those expected in the school-wide population (Horvitz and Thompson, 1952). We report all results adjusted in this way, rather than crude rates or averages. Response rates for the various racial/ethnic groups were as follows (Sexton et al., 2003): Whites (71%), African-Americans (35%), Hispanics (80%), Somalis (66%), Asians (64%), Others (45%).

Our recruitment yielded 153 participants, of whom 136 (89%) satisfactorily completed both the health questionnaire and lung spirometry. Distribution by race and ethnicity (Table 1) showed African-Americans, Somalis, and Hispanics were the largest groups, with smaller numbers of Asians (8 Cambodians, 4 Laotians), Whites (11) and Others (4).

Only half of the families spoke English as the primary language at home, emphasizing the high proportion of immigrant families included in this study. Measures of socioeconomic status indicated low median household incomes in each racial/ethnic group, and low educational levels in that only 53.1% of all families had at least one parent who graduated from high school or equivalent (Table 1). Further evidence of poverty was reflected by more than 80% of children at these two schools who qualified for free school meals.

The overall rates for tobacco smoking in the home among primary caregivers was 23.3%, and for anyone in the home was 31.6%; African-Americans, Asians and White/Others had the highest percentages of homes with at least one smoker, while Somalis and Hispanics had fewer homes with a smoker (Table 1).

#### 3.1. Symptoms

Respiratory illness and symptom rates differed significantly among the racial/ethnic groups (Table 2). Of particular interest were children whose parents reported the child had physician-diagnosed asthma, whether the

Table 1  
Racial, ethnic, socioeconomic, and demographic characteristics of poor, inner-urban, Minneapolis schoolchildren<sup>a</sup>

	African-American	Somali immigrant	Hispanic	Asian	White/other	Total
Number	32 (35.7%)	34 (18.2%)	43 (23.8%)	12 (8.5%)	15 (13.8%)	136 (100%)
Females	17 (48.2%)	17 (52.1%)	17 (38.4%)	6 (45.2%)	7 (36.4%)	64 (44.7%)
Males	15 (51.8%)	17 (47.9%)	26 (61.6%)	6 (54.8%)	8 (63.6%)	72 (55.3%)
Attend Lyndale School	18 (45.4%)	16 (58.3%)	7 (20.4%)	12 (100%)	13 (86.2%)	66 (52.1%)
English spoken at home	32 (100%)	0	2 (6.2%)	0	12 (84.2%)	46 (48.7%)
Median household income, \$	25,000	<10,000	15,000	<10,000	25,000	15,000
One or more parents graduated HS or equivalent	30 (95.8%)	8 (18.0%)	2 (7.0%)	3 (24.7%)	12 (84.2%)	55 (53.1%)
Primary caregiver smokes	15 (46.6%)	0	3 (8.9%)	1 (10.4%)	5 (26.8%)	29 (21.4%)
Any smoker present in the home	17 (54.2%)	0	8 (19.1%)	4 (39.4%)	6 (31.5%)	40 (31.1%)
Mean age (yr) (SD)	9.3 (1.5)	9.4 (1.0)	8.8 (0.9)	9.2 (1.3)	9.2 (1.4)	8.8 (1.2)
Mean height (cm) (SD)	138.1 (11.7)	143.5 (8.7)	133.8 (7.8)	134.2 (8.2)	134.4 (10.1)	137.2 (9.9)
Mean weight (kg) (SD)	38.6 (15.4)	36.5 (8.1)	36.8 (9.9)	30.3 (6.4)	33.3 (14.1)	36.4 (11.4)
Mean BMI, kg/m <sup>2</sup> (SD) <sup>b</sup>	19.8 (4.0)	17.6 (3.3)	20.2 (4.2)	16.7 (2.1)	18.0 (4.5)	19.0 (4.1)

<sup>a</sup>All proportions (percentages) and means were calculated using weighted counts that adjusted for the sampling and response probabilities.

<sup>b</sup>Body Mass Index (BMI) = Weight (kg)/Height<sup>2</sup> (m).

Table 2  
Weighted rates<sup>a</sup> of parent-reported respiratory symptoms, asthma, and related disorders

	African-American	Somali immigrant	Hispanic	Asian	White/other	Total
Asthma <sup>b</sup>	8 (25.9%)	1 (1.8%)	4 (9.3%)	0	3 (25.8%)	16 (15.4%)
Current asthma (≥1 episode in last 12 months)	7 (24.1%)	1 (1.8%)	2 (4.5%)	0	3 (25.8%)	13 (13.6%)
Child takes medication for asthma	6 (16.4%)	1 (1.8%)	1 (3.1%)	0	3 (25.8%)	11 (10.5%)
Cough apart from colds	8 (25.6%)	1 (3.4%)	1 (2.7%)	1 (10.4%)	5 (30.6%)	16 (15.5%)
Phlegm apart from colds	2 (5.2%)	0	3 (7.8%)	0	1 (9.6%)	6 (6.1%)
Wheeze apart from colds	3 (7.4%)	0	1 (1.5%)	0	3 (25.8%)	11 (13.7%)
Wheeze most days or nights	2 (5.2%)	1 (3.4%)	1 (1.5%)	0	1 (9.6%)	10 (12.1%)
≥1 Attack of wheezing and breathlessness	9 (29.8%)	1 (1.8%)	1 (3.1%)	0	4 (42.3%)	15 (17.5%)
Eczema <sup>b</sup>	3 (5.1%)	1 (1.8%)	1 (2.3%)	0	2 (16.3%)	7 (5.2%)
Allergic reaction to dust or pollen <sup>b</sup>	2 (9.0%)	2 (8.1%)	1 (2.7%)	0	4 (37.0%)	9 (10.4%)
Bronchitis <sup>b</sup>	4 (10.3%)	4 (12.7%)	6 (15.6%)	0	2 (26.5%)	16 (13.4%)
Pneumonia <sup>b</sup>	1 (1.8%)	0	2 (4.7%)	2 (17.6%)	1 (10.0%)	6 (4.6%)
Hospitalized for a chest illness before age 2 yr	5 (10.1%)	0	5 (13.7%)	3 (29.2%)	0	13 (10.4%)

<sup>a</sup>All proportions (percentages) were calculated using weighted counts that adjusted for the sampling and response probabilities.

<sup>b</sup>Condition diagnosed by a physician and reported by a parent or guardian of the child.

child still had asthma, and whether the child was taking any medication for asthma at the time of the interview.

The adjusted rate of physician-diagnosed, lifetime asthma for all children was 15.4% (95% confidence interval, 95CI, 9.3–21.5%), ranging from zero for Asians to nearly 26% among African-Americans and White/Others (Table 2); the rates of physician-diagnosed asthma differed significantly among the various groups (Chi-square = 15.2;  $P < 0.01$ ). The adjusted rate for children who had at least one asthmatic episode in the last year (current asthma) was 13.6% (95CI 7.8–19.4%), while 10.5% (5.3–15.7%) were taking some form of asthma medication at the time of the interview (Table 2). The adjusted rate for one or more attacks of wheezing and breathlessness was 17.5% (11.1–23.9%) and differed significantly between the racial/ethnic groups ( $P < 0.01$ ), ranging from zero for Asians to 42.3% for White/Others (Table 2).

Adjusted rates for parent-reported, physician-diagnosed eczema (5.2%, 1.5–8.9%) and allergic reactions to pollen or dust (10.4%, 5.3–15.5%) showed wide variability among the racial/ethnic groups, with White/Others having the highest rates for both.

### 3.2. Lung function

Mean lung function showed expected differences between males and females of the same race/ethnicity (Table 3). Individuals' values for FEV<sub>1</sub>, FVC were compared with reference data for White children (Knudson et al., 1983), and expressed as percentages of the respective predicted values (Table 3). Overall, FEV<sub>1</sub> averaged 89.5%-predicted (95CI 87.3–91.7%) and FVC averaged 90.9%-predicted (88.2–93.6%). Whites/Others had mean values for FEV<sub>1</sub> and FVC of 100.5 and 100.7%-predicted, indicating close agreement on average with the prediction equations of



Table 3

Weighted mean (SD) values<sup>a</sup> for lung function among poor, inner-urban schoolchildren compared with data of Knudson et al. (1983)

	African-American	Somali immigrant	Hispanic	Asian	White/other	Total
FEV <sub>1</sub> (L)						
Males ( <i>N</i> = 72)	1.68 (0.32)	1.79 (0.40)	1.77 (0.33)	1.77 (0.40)	1.85 (0.46)	1.76 (0.35)
Females ( <i>N</i> = 64)	1.58 (0.37)	1.71 (0.48)	1.74 (0.45)	1.37 (0.43)	1.74 (0.21)	1.66 (0.42)
FVC (L)						
Males	1.98 (0.45)	2.02 (0.50)	2.09 (0.52)	2.02 (0.56)	2.15 (0.40)	2.05 (0.50)
Females	1.80 (0.49)	1.88 (0.56)	2.03 (0.46)	1.46 (0.43)	1.97 (0.36)	1.87 (0.49)
FEV <sub>1</sub> /FVC (%)	85.6% (7.1)	90.4% (8.5)	85.5% (8.5)	91.5% (5.0)	88.8% (7.1)	87.4% (7.6)
FEV <sub>1</sub> , percent-predicted <sup>b</sup>	86.5% (11.5)	81.5% (12.7)	94.5% (10.2)	87.5% (12.3)	100.5% (10.4)	89.5% (12.9)
FVC, percent-predicted <sup>b</sup>	89.4% (14.8)	79.1 (14.1)	98.5% (12.9)	85.2% (14.2)	100.7% (12.9)	90.9% (15.8)

<sup>a</sup>All means and standard deviations (SD) were calculated using weighted values that adjusted for the sampling and response probabilities.<sup>b</sup>Predicted values were obtained from equations of Knudson et al. (1983) for children 6–12 years old.

Table 4

Coefficients from lung function models showing effects of demographic variables, gender, school attended, and racial or ethnic group in poor inner-urban school children

	FEV <sub>1</sub>	ln(FEV <sub>1</sub> )	FVC	ln(FVC)
Intercept	***−2.378 (0.277)	***−11.908 (0.984)	***−3.240 (0.348)	***−13.126 (1.061)
Height, cm	*** 0.076 (0.007)	*** 0.097 (0.009)		
ln(Height)	*** 2.471 (0.242)	*** 2.730 (0.261)		
Age (yr)	−0.005 (0.022)	−0.013 (0.027)		
ln(Age)	−0.057 (0.124)	−0.006 (0.134)		
BMI (kg/m <sup>2</sup> )	0.007 (0.006)	**0.018 (0.007)		
ln(BMI)	0.099 (0.066)	* 0.174 (0.071)		
Asthma	−0.003 (0.065)	−0.027 (0.040)	0.026 (0.081)	−0.001 (0.043)
Female	***−0.129 (0.039)	***−0.090 (0.024)	***−0.207 (0.050)	***−0.113 (0.026)
Whittier School	**0.131 (0.046)	**0.090 (0.029)	**0.187 (0.058)	*** 0.113 (0.031)
African-American	***−0.281 (0.071)	***−0.180 (0.044)	***−0.329 (0.089)	***−0.184 (0.047)
Somali	***−0.386 (0.076)	***−0.259 (0.046)	***−0.512 (0.095)	***−0.296 (0.050)
Hispanic	*−0.177 (0.075)	**−0.129 (0.046)	*−0.201 (0.095)	*−0.121 (0.050)
Asian	−0.156 (0.087)	*−0.119 (0.053)	−0.201 (0.095)	*−0.133 (0.057)
Model <i>R</i> <sup>2</sup>	0.698	0.692	0.717	0.714
Adjusted <i>R</i> <sup>2</sup>	0.674	0.667	0.694	0.691

Statistical notations for significance of coefficient differing from zero: \**P* < 0.05; \*\*0.001 < *P* < 0.01; \*\*\**P* < 0.001.

Knudson, but all other groups had mean percent-predicted values significantly less than 100% (Table 3).

Lung function was analyzed further by multiple linear regression. Independent variables were included for age, height, body mass index (BMI), physician-diagnosed asthma, female gender, school attended, and the various racial/ethnic groups. A simple linear model and a logarithmic model were examined (Table 4); each model had *R*<sup>2</sup> and adjusted-*R*<sup>2</sup> values greater than 0.66. Height, female gender, Whittier School, and the racial/ethnic variables were consistently significant in models for FEV<sub>1</sub>. The same variables plus BMI were significant predictors for FVC. African-American, Somali, Hispanic, and Asian children had significantly smaller values of FEV<sub>1</sub> and FVC than White/Others. Gender differences in lung function were demonstrated also, with girls having smaller FEV<sub>1</sub> and FVC than boys for a given age, height, BMI, racial/ethnic group, and school attended. The

contributions of age and asthma terms were small and not statistically significant (Table 4).

Coefficients obtained for the indicator variables (1,0) that adjusted for asthma, school, gender, and racial/ethnic groups in the logarithmic models were used to calculate the ratio of effect (multiplying factor) attributable to having physician-diagnosed asthma, attending Whittier School, being female, or belonging to one of the designated racial/ethnic groups (Table 5). For the various racial/ethnic groups, these multiplying factors are analogous to percent-predicted values relative to Whites/Others after adjusting for gender and the other covariates in the regression models. The lowest lung function occurred in Somalis whose FEV<sub>1</sub> and FVC values were 77.2% and 74.4%, respectively, relative to White/Others; African-Americans (83.5% and 83.2%) were the next lowest followed by Hispanics (87.9%, 88.6%) and Asians (88.8%, 87.5%). Females had FEV<sub>1</sub> and FVC values that were 91.0% and

89.3%, respectively, of the values in males of the same age, height, BMI, school, and race/ethnicity. Attendees at Whittier School had 9.4% greater FEV<sub>1</sub> values and 12.0% greater FVC values than attendees at Lyndale School. Physician-diagnosed asthma was not associated significantly with lung function.

### 3.3. Atopy

Blood levels of total and allergen-specific IgE were measured in 97 children (Table 6). The distributions of IgE levels were skewed and data are presented for the medians, 10th and 90th percentiles. In the laboratory performing these tests, an increased level of total IgE is regarded as a value greater than 100 IU/mL, and an increased level of specific IgE is greater than 0.35 IU/mL.

Table 5

Multiplying factors for FEV<sub>1</sub> and FVC obtained from logarithmic regression models that included variables for asthma, Whittier school, female gender, and specific racial or ethnic groups<sup>a</sup>

	FEV <sub>1</sub> (L)	FVC (L)
Physician-diagnosed asthma	0.973	0.999
Attendance at Whittier School	**1.094	***1.120
Female gender	***0.914	***0.893
African-American	***0.835	***0.832
Somali	***0.772	***0.744
Hispanic	**0.879	*0.886
Asian	*0.888	*0.875

Statistical notations for values differing significantly from 1.0: \* $P < 0.05$ ; \*\* $0.001 < P < 0.01$ ; \*\*\* $P < 0.001$ .

<sup>a</sup>Reference group is White/Other males of the same age, height, and BMI who did not have physician-diagnosed asthma and attended Lyndale school.

Table 6

Total and specific serum IgE levels (IU/ml) for different racial/ethnic groups<sup>a</sup>

	African-American	Somali immigrant	Hispanic	Asian	White/other	Total
Number (% of ethnic group)	18 (57.0%)	29 (86.0%)	31 (67.8%)	7 (63.6%)	12 (81.0%)	97 (68.7%)
Total IgE, (IU/mL) <sup>b</sup>	28.9 (6.0, 147)	79.8 (13.6, 746)	53.4 (7.1, 299)	213.6 (33.2, 645)	173.7 (76.7, 1164)	76.7 (7.1, 501)
Number (%) > 100 (IU/mL)**	4 (28.4%)	13 (39.3%)	11 (32.4%)	4 (54.8%)	8 (73.7%)	40 (41.3%)
<i>Specific IgE (IU/mL)</i>						
Mite 1 ( <i>D. pteronyssinus</i> ) <sup>b</sup>	0.21 (ND,1.54)	0.19 (ND,3.55)	0.17 (0.08,0.50)	0.80 (ND,11.93)	1.56 (ND,5.78)	0.21 (ND,3.29)
Number (%) > 0.35 (IU/mL)*	6 (23.5%)	7 (29.2%)	5 (18.3%)	4 (56.3%)	8 (55.5%)	30 (34.7%)
Mite 2 ( <i>D. farinae</i> ) <sup>b</sup>	0.04 (ND,0.62)	0.08 (ND,0.15)	0.12 (0.02,0.21)	0.08 (0.01,2.57)	0.02 (ND,0.64)	0.08 (ND,0.62)
Number (%) > 0.35 (IU/mL)	4 (26.5%)	0	1 (2.3%)	1 (18.2%)	2 (13.5%)	8 (12.0%)
Cockroach <sup>b</sup>	0.17 (0.04,0.41)	0.10 (ND,1.99)	0.36 (0.13,2.47)	0.06 (ND,1.16)	0.11 (ND,2.00)	0.18 (ND,2.00)
Number (%) > 0.35 (IU/mL)*	4 (31.0%)	11 (29.9%)	16 (52.3%)	1 (11.1%)	3 (22.8%)	35 (32.8%)
Cat Dander <sup>b</sup>	0.05 (ND,0.63)	0.16 (ND,1.34)	0.45 (0.08,2.53)	0.02 (0.01,0.58)	0.19 (0.04,1.71)	0.21 (ND,1.34)
Number (%) > 0.35 (IU/mL)*	3 (24.0%)	11 (33.2%)	19 (62.1%)	2 (34.6%)	2 (17.1%)	37 (34.7%)
Ragweed <sup>b</sup>	0.11 (0.02,1.46)	0.23 (ND,0.75)	0.24 (0.13,1.14)	0.04 (ND,7.83)	0.57 (0.01,8.17)	0.20 (0.02,1.46)
Number (%) > 0.35 (IU/mL)*	5 (34.9%)	8 (30.0%)	10 (33.7%)	2 (29.3%)	8 (55.8%)	33 (36.4%)

Abbreviations: IU/mL = International Units per milliliter. ND = Not Detected.

Significance of Mantel-Haenszel Chi-square tests for differences between groups: \* $P < 0.05$ ; \*\* $P < 0.01$ .

<sup>a</sup>All proportions, percentiles, Chi-square statistics, and  $P$ -values were adjusted for selection and response probabilities.

<sup>b</sup>Values are the medians (10th, 90th percentiles) for each racial/ethnic group. Statistical notations for each racial/ethnic group.

The overall adjusted rate for increased total IgE levels (>100 IU/mL) was 41.3% (95CI 33.0–49.6%), with rates among the various racial/ethnic groups ranging from 28.4% in African-Americans to 73.7% for White/Others (Table 6). The adjusted overall rates for increased specific IgE levels (>0.35 IU/mL) were in the range of 32–36%, with the exception of *D. farinae* which was increased in only 12.0% of the population.

Significant differences in specific IgE levels were found among the various racial/ethnic groups (Table 6). IgE levels to house dust mite (*D. pteronyssinus*) were particularly increased among Asians (56.3%) and White/Others (55.5%) relative to the other groups. Cockroach-specific IgE was highest in Hispanics (52.3%), and IgE levels to ragweed were highest among White/Others (55.8%). IgE to cat dander allergy was particularly increased among Hispanics (62%) but only two of the Hispanic homes had cats. Among Somalis, whose homes had no cats, one-third of children had increased IgE levels to cat dander.

As a measure of atopy, children were identified who had either a total IgE greater than 100 IU/mL or an allergen-specific IgE level greater than 0.35 IU/mL. Of those children so defined, the racial/ethnic group with the highest percentage of atopy was White/Others (100%), followed by Asians (81.8%), Somalis (78.1%), Hispanics (77.2%), and African-Americans (66.6%). Differences in the rates of atopy among the various groups was significant ( $P < 0.01$ ).

## 4. Discussion

The main goals of the SHIELD study were to assess the environmental exposures and associated health outcomes among poor, inner-urban Minneapolis schoolchildren. A secondary goal was to examine the rates of respiratory

symptoms and illnesses and the determinants of lung function in the various racial/ethnic groups. This paper provides baseline information about respiratory symptoms, lung function, and atopy in relation to age, gender, school attended, and racial/ethnic group.

The numbers of children included were limited by a study design that emphasized the collection of many environmental samples, measurement of a large number of exposure biomarkers, and total cost. As a result the numbers of students in each racial/ethnic group were small, making interpretation of the present findings difficult. Nevertheless, large differences in respiratory health were observed among the racial/ethnic groups, and sufficient statistical power was present to analyze some of those differences. The data were adjusted consistently to describe the expected experience for the total population or the respective racial/ethnic groups attending each school.

The parent-reported, adjusted rate for lifetime asthma prevalence in the two schools (15.4%) was similar to the national estimate of 14.0% for children aged 5–11 years obtained in the 2003 National Health Interview Survey (NHIS) (Dey and Bloom, 2005). However, the adjusted rate for those who experienced one or more asthma attacks in the last year (13.6%, 95%CI 7.8–19.4%) was more than twice the reported national rate of 5.9% (SE 0.4%) for annual asthma among children aged 5–11 years (Dey and Bloom, 2005). Although NHIS used slightly different questions to determine annual asthma prevalence, this difference cannot explain the much higher rate of ongoing asthma observed in the present study. Moreover, 10.5% of children in our two schools were currently taking asthma medications, another indication of persistent asthma, that again exceeded the national estimate for annual asthma prevalence.

Although the participants in the present study came from families living in the same inner-urban neighborhoods, substantial differences in respiratory health were apparent between the various racial/ethnic groups. Children from the predominantly long-resident, English-speaking African-American and White/Other groups had the highest rates of physician-diagnosed asthma, eczema, and allergic reactions to dust or pollen, and also reported the highest rates of tobacco smoking by the child's primary caregiver.

Asthma and allergy rates among the recently arrived immigrant groups of Somali and Asian children were substantially lower than the long resident groups, while Hispanic children (whose resident status was unknown) were intermediate between long residents and recent immigrants. The finding of lower asthma rates among immigrant children is consistent with data reported for immigrant Hispanic children to the United States (Eldeirawi et al. 2005) and for Asian immigrants in Australia (Leung et al. 1994).

National statistics (Dey and Bloom, 2005; Mannino et al., 1998, 2002) and other findings in the US (Malveaux and Fletcher-Vincent, 1995) demonstrate that the highest

rates of asthma in the US occur among non-Hispanic black children. The rates of lifetime and ongoing asthma for American-born black children (African-Americans) in this study were also among the highest of the various racial/ethnic groups. Unlike some previous studies, however, African-American children attending these schools came from families that were better educated and had higher median incomes than most of the other racial/ethnic groups living in this inner-urban neighborhood (Table 1). It should be noted too that the participation rate of African-American families in the present study was 35%, the lowest of all groups (Sexton et al., 2003), so the observed asthma rates may not have been representative of all African-American children who attended the schools.

Lifetime asthma was infrequent among the immigrant Somali and Asian children (combined prevalence 2.1%). Rates of asthma or other respiratory disorders have not been reported from their respective host countries, so it is unclear whether the low prevalence pertains to experiences before or after the families arrived in the US. Certainly, data on asthma rates vary widely among different countries, and appear to reflect many possible factors, including hygiene, diet, cigarette smoking, traffic pollution, antenatal exposures, physical activity and obesity (Gold and Wright, 2005). The present study was not designed to identify behavioral or lifestyle risk factors that might help distinguish between racial/ethnic rates of asthma, but it is apparent that the highest rates of asthma occurred in children from racial/ethnic groups that had the highest rates of smoking among primary caregivers (Table 1). An index of obesity (BMI) did not differ widely among the various racial/ethnic groups (Table 1). High rates of atopy were present in all racial/ethnic groups, even the Somalis and Asians (Table 6), suggesting that the presence of atopy was a poor predictor overall for asthma.

#### 4.1. Lung function

Lung function tests of African-Americans and immigrant racial/ethnic groups differed significantly from White/Others. Other investigators have noted similar differences for African-American and Hispanic children (see, for example, Schwartz et al., 1988; Hsi et al., 1983; Hsu et al., 1979), but data for Somali and Cambodian/Lao children are lacking.

Coefficients obtained in this study for girls and for African-American children using the log-transformed lung function models were compared with analyses performed by Schwartz and coworkers (1988) who used a similar logarithmic model for a much larger sample of children in the NHANES II national survey. Schwartz found that girls had FEV<sub>1</sub> and FVC that were 3.5% and 5.8% less than the respective values for boys; these compare with values of 8.8% and 11.0% for our study. Schwartz also found that African-American children had FEV<sub>1</sub> and FVC values that were 11.4% and 12.6%, respectively, less than values for white children. Again, data from the present study indicate

larger differences, with African-Americans having values 16.5% and 15.9% less than White/Others. Hsu and coworkers (1979) and Hsi and colleagues (1983) measured lung function in Mexican-American, African-American, and White school children aged 7–20 years, and found that both Hispanic and African-American children had lower values of lung function than Whites for a given standing height and gender.

The lung function findings reported here for immigrant Somali and Cambodian/Lao children are the first such findings for these groups in the United States, and no lung function data have been reported from their respective home countries. Lung function data have been reported for other Asian groups, including Chinese children in Hong Kong (Ip et al., 2000a b), Singapore (Connett et al., 1994), and Taiwan (Hsieh and Shen, 1988); for Indian children in Britain (Chinn and Rona, 1992), India (Malik and Jindal, 1985), and Singapore (Connett et al., 1994); and for Malay children in Malaysia (Azizi and Henry, 1994) and Singapore (Connett et al., 1994). Children from these Asian populations have smaller lung volumes than Caucasians for a given age, height, and gender. The present finding of smaller lung volumes for Cambodian and Lao children relative to Whites is consistent, therefore, with lung function findings reported for other Asian children.

More recent findings reported by Ip and colleagues (2000a, b) among Chinese children aged 7–19 years in Hong Kong emphasize that the relationships of lung function and lung volumes to age and height may increase over time within a particular racial group, and that such increases likely reflected “secular change in body size, height at puberty, and [improved] overall health in the pediatric population in Hong Kong” between 1985 and 1996. Furthermore, “exogenous factors may contribute significantly to differences in lung function values among ethnic groups and it is important to examine normative values of various populations for secular trends” (Ip et al., 2000a). These observations may be relevant to our Somali and Asian children who were from poor, immigrant refugee families, and likely experienced under nutrition and other privations associated with poverty in a developing country.

#### 4.2. Atopy

It is noteworthy that IgE levels for different allergens varied among the racial/ethnic groups: the highest IgE levels for house dust mite were among Asians and White/Others, with more than half of the children in each group having increased levels of specific IgE for *D. pteronyssinus*, while more than half the Hispanic children had increased levels of IgE for cockroaches and cat dander. When we examined children’s homes for allergens, significantly higher levels of dust mite allergens ( $> 2 \mu\text{g/g}$  of dust) were found in Asian and White/Other homes relative to other racial/ethnic groups, and Hispanic homes were among

several groups that had significantly higher levels of cockroach allergen ( $> 2 \mu\text{g/g}$  of dust) (Adgate, unpublished findings).

With regard to sensitization to cat dander among Hispanic children, only two Hispanic homes kept cats, which does not explain the high rate of sensitization among Hispanic children. Cat allergen is unusual, however, in that it remains suspended in air for long periods and can be transported on clothing from one environment to another (Bollinger et al., 1996; IOM, 2000). Children have become sensitized without having a cat in the home, simply by coming in contact with cat dander in another environment (Bollinger et al., 1996). A similar mode of sensitization may have occurred among children in the present study. Of note is the fact that 81% of families included in this study were living in rented accommodation (Adgate et al., 2004), and a substantial fraction moved residence each year (Sexton et al., 2003). It was also possible that households may have had cats previously, but got rid of them when a child developed allergic symptoms.

Levels of the various allergens were examined also in dust samples from the two schools that these children attended and school levels were low relative to levels found in the homes (Adgate, unpublished findings). Based on the allergen levels found in the homes and schools, it seems likely that environmental exposures in the home explain the different levels of specific IgE among the various racial/ethnic groups of children in this study.

#### 4.3. Differences between the schools

Several respiratory health differences were found among children in relation to the school attended, with those at the Lyndale School having generally lower levels of lung function. Students at Lyndale also had higher levels of specific IgE to dust mite, while those at Whittier had higher levels of specific IgE to cat dander and cockroach antigen. To a large extent the findings for specific IgE levels reflected the racial/ethnic differences between students attending the two schools, with all of the immigrant Asians and most of the White/Others being found at Lyndale, and the substantial majority of Hispanics attending the Whittier School.

Differences in lung function, however, cannot be explained as readily because racial/ethnic terms were used in the regression analyses for FEV<sub>1</sub> and FVC, but school-related differences in lung function persisted. This may have reflected some residual confounding between School and Race/Ethnicity, despite including terms for each in the regression models. While the reason for this difference between schools was unclear, it did not seem to be related to any of the airborne agents or contaminants that were measured in the schools (Adgate, unpublished findings).



## 5. Conclusions

Despite the relatively small numbers of children studied, we found substantial ethnic/racial differences in asthma prevalence, specific IgE levels, and lung function among poor inner-urban schoolchildren. Overall asthma prevalence was high in non-immigrant children, with much lower rates among immigrant children from Africa and Asia. Similar differences between immigrant and resident populations have been noted by others (for example, Dey and Bloom; 2005, Gold and Acevedo-Garcia, 2005; Leung, 1994).

Specific circulating IgE levels varied among the racial/ethnic groups, largely reflecting different allergen exposures occurring in the home. Lung function data demonstrated gender- and race-related differences, consistent with previous findings. School environments did not appear to contribute to the observed rates of asthma or atopy, but unexplained differences in lung function were associated with attendance at the two schools.

The present findings emphasize that children's asthma rates can vary widely in poor, inner-urban neighborhoods, and that recent immigration and race/ethnicity are important predictors for unusually low or high asthma rates. The relationship of childhood asthma to poverty and inner-urban living is complex. Among areas deserving of further study are the indoor exposures and respiratory health of immigrant and non-immigrant children living in similar poor urban environments. Inner urban groups in Minneapolis have substantial immigrant populations and appear well suited to examining the health differences between immigrant and resident populations. Such studies may help to elucidate further the perplexing increase in childhood asthma that has been occurring in industrialized countries.

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## References

Adgate, J.L., Church, T.R., Ryan, A.D., Ramachandran, G., Fredrickson, A., Morandi, M.T., et al., 2004. Outdoor, indoor, and personal exposure to VOCs in children. *Environ. Health Perspect.* 112, 1386–1392.

- Anderson, H.R., Bailey, P.A., Cooper, J.S., Palmer, J.C., West, S., 1983. Morbidity and school absence caused by asthma and wheezing illness. *Arch. Dis. Child.* 58, 777–784.
- Anderson, H.R., 1989. Increase in hospital admissions for childhood asthma: trends in referral, severity, and readmissions from 1970 to 1985 in health region of the United Kingdom. *Thorax* 44, 614–619.
- ATS (American Thoracic Society), 1987. Standardization of spirometry—1987 update. *Am. Rev. Respir. Dis.* 136, 1285–1298.
- Azizi, B.H.O., Henry, R.L., 1994. Ethnic differences in normal spirometric function of Malaysian children. *Respir. Med.* 88, 349–356.
- Bascom, R., Bromberg, P.A., Costa, D.A., et al., 1996. State of the art: health effects of outdoor air pollution, part II. *Am. J. Respir. Crit. Care Med.* 153, 477–498.
- Beasley, R., Pearce, N., Crane, J., 1997. International trends in asthma mortality. In: Chadwick, D., Cardow, G. (Eds.), *The Rising Trends in Asthma*. Ciba Foundation Symposium 206. Wiley, Chichester, pp. 140–156.
- Bollinger, M.E., Eggleston, P.A., Flanagan, E., Wood, R.A., 1996. Cat antigen in homes with and without cats may induce allergic symptoms. *J. Allergy Clin. Immunol.* 97, 907–914.
- Chinn, S., Rona, R.J., 1992. Height and age adjustment for cross sectional studies of lung function in children aged 6–11 years. *Thorax* 47, 707–714.
- Connett, G.J., Quak, S.H., Wong, M.L., Teo, J., Lee, B.W., 1994. Lung function reference values in Singaporean children aged 6–18 years. *Thorax* 49, 901–905.
- Dey, A.N., Bloom, B., 2005. Summary health statistics for US children: National Health Interview Survey, 2003. National Center for Health Statistics. *Vital Health Stat.* 10 (223).
- Eldeirawi, K., McConnell, R., Freels, S., Persky, V., 2005. Associations of place of birth with asthma and wheezing in Mexican American children. *J. Allergy Clin. Immunol.* 116, 42–48.
- EPA (US Environmental Protection Agency), 2001. National Air Quality and Emissions Trends Report, 1999. EPA 454/R-01-004. Research Triangle Park, NC: USEPA.
- Ferris, B.G., 1978. Epidemiology Standardization Project. *Am. Rev. Respir. Dis.* 118 (part 2), 36–51.
- Gold, D.R., Acevedo-Garcia, D., 2005. Immigration to the United States and acculturation as risk factors for allergy and asthma. *J. Allergy Clin. Immunol.* 116, 38–41.
- Gold, D.R., Wright, R., 2005. Population disparities in asthma. *Ann. Rev. Pub. Health* 26, 89–113.
- Halfon, N., Newacheck, P.W., 1993. Childhood asthma and poverty: differential impacts and utilization of health services. *Pediatrics* 91, 59–61.
- Hecht, S.S., Ye, M., Carmella, S.G., Fredrickson, A., Adgate, J.L., Greaves, I.A., et al., 2001. Metabolites of a tobacco-specific lung carcinogen in the urine of elementary school-aged children. *Cancer Epidemiol. Biomarkers Prev.* 10, 1109–1116.
- Horvitz, D., Thompson, D., 1952. A generalization of sampling without replacement from a finite universe. *J. Am. Stat. Assoc.* 47, 663–685.
- Hsi, B.P., Hsu, K.H.K., Jenkins, D.E., 1983. Ventilatory functions of normal children and young adults: Mexican-American, white, and black. III. Sitting height as a predictor. *J. Pediatr.* 102, 860–865.
- Hsieh, K.H., Shen, J.J., 1988. Prevalence of childhood asthma in Taipei, Taiwan and other Asia Pacific countries. *J. Asthma* 25, 73–82.
- Hsu, K.H.K., Hsi, B.P., Thompson, V., Tanakawa, N., Hsieh, G.S.J., 1979. Ventilatory functions of normal children and young adults: Mexican-American, white, and black. I Spirometry. *J. Pediatr.* 95, 12–23.
- IOM (Institute of Medicine), 2000. *Clearing the Air: Asthma and Indoor Air Exposures*. National Academy Press, Washington, DC.
- Ip, M.S.M., Karlberg, E.M., Chan, K.-N., Karlberg, J.P.E., Luk, K.D.K., Leong, J.C.Y., 2000a. Lung function reference values in Chinese children and adolescents in Hong Kong. II. Prediction equations for plethysmographic lung volumes. *Am. J. Respir. Crit. Care Med.* 162, 430–435.

- Ip, M.S.M., Karlberg, E.M., Karlberg, J.P.E., Luk, K.D.K., Leong, J.C.Y., 2000b. Lung function reference values in Chinese children and adolescents in Hong Kong. I. Spirometric values and comparison with other populations. *Am. J. Respir. Crit. Care Med.* 162, 424–429.
- ISAAC (The International Study of Asthma and Allergies in Childhood Steering Committee), 1998. Worldwide variation in prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and atopic eczema: ISAAC. *Lancet* 351, 1225–1232.
- Jackola, D.R., Pierson-Mullany, L.K., Liebler, C.L., Blumenthal, M.N., Rosenberg, A., 2002. Variable binding affinities for allergen suggest a 'selective competition' among immunoglobulins in atopic and non-atopic humans. *Mol. Immunol.* 1178, 1–11.
- Kheradman, F., Rishi, K., Corry, D.B., 2002. Environmental contributions to the allergic asthma epidemic. *Environ. Health Perspect.* 110 (suppl4), 553–556.
- Knudson, R.J., Lebowitz, M.D., Holberg, C.J., Burrows, B., 1983. Changes in the normal maximal expiratory flow-volume curve with growth and aging. *Am. Rev. Respir. Dis.* 127, 725–734.
- Koenig, J.Q., 1999. Air pollution and asthma. *J. Allergy Clin. Immunol.* 104, 717–722.
- Leung, R., 1994. Asthma, allergy and atopy in South-east Asian immigrants in Australia. *Aust. NZ J. Med.* 24, 255–257.
- Leung, R.C., Carlin, J.B., Burdon, J.G., Czarny, D., 1994. Asthma, allergy and atopy in Asian immigrants in Melbourne. *Med. J. Aust.* 161, 418–425.
- Leikauf, G.D., 2002. Hazardous air pollutants and asthma. *Environ. Health Perspect.* 110 (suppl 4), 505–526.
- Lozano, P., Sullivan, S.D., Smith, D.H., Weiss, K.B., 1999. The economic burden of asthma in US children: estimates from the National Medical Expenditure Survey. *J. Allergy Clin. Immunol.* 104, 957–963.
- Malik, S.K., Jindal, S.K., 1985. Pulmonary function in healthy children. *Indian Pediatr.* 22, 677–681.
- Malveaux, F.J., Fletcher-Vincent, S.A., 1995. Environmental risk factors of childhood asthma in urban centers. *Environ. Health Perspect.* 103 (suppl 6), 59–62.
- Mannino, D.M., Homa, D.M., Pertowoki, C.A., Ashizawa, A., Nixon, L.L., Johnson, C.A., et al., 1998. CDC: Surveillance for Asthma—United States, 1960–1995. *MMWR* 47 (SS-1), 1–27.
- Mannino, D.M., Homa, D.M., Akinbami, L.J., Moorman, J.E., Gwynn, C., Redd, S.C., 2002. CDC: Surveillance for Asthma—United States, 1980–1999. *MMWR* 51 (SS-1), 1–13.
- Newacheck, P.W., Halfon, N., 2000. Prevalence, impact, and trends in childhood disability due to asthma. *Arch. Pediatr. Adolesc. Med.* 154, 287–293.
- Patterson, W., Werness, P., Payne, W.J., Mattsos, P., Leflar, C., Melander, T., et al., 1994. Random and continuous-access immunoassays with chemiluminescent detection by ACCESS Automated analyzer. *Clin. Chem.* 40, 2042–2045.
- Pearce, N., Weiland, S.K., Keil, U., Langridge, P., Anderson, H.R., Strachan, D., Bauman, A., 1993. Self-reported prevalence of asthma in children in Australia, England, Germany and New Zealand: an international comparison using the ISAAC protocol. *Eur. Respir. J.* 6, 1455–1461.
- Rasmussen, F., Taylor, D.R., Flannery, E.M., Cowan, J.O., Greene, J.M., Herbison, P., Sears, M.R., 2002. Risk factors for hospital admission for asthma from childhood to young adulthood: A longitudinal population study. *J. Allergy Clin. Immunol.* 110, 220–227.
- Redd, S.C., 2002. Asthma in the United States: burden and current theories. *Environ. Health Perspect.* 110 (suppl 4), 557–560.
- Schwartz, J.D., Katz, S.A., Fegley, R.W., Tockman, M.S., 1988. Analysis of spirometric data from a national sample of healthy 6- to 24-year-olds (NHANES II). *Am. Rev. Respir. Dis.* 138, 1405–1414.
- Sexton, K., Greaves, I.A., Church, T.R., Adgate, J.L., Ramachandran, G., Tweedie, R., et al., 2000. A school-based strategy to assess children's environmental exposures and related health effects in economically disadvantaged urban communities. *J. Exp. Anal. Environ. Epidemiol.* 10, 682–694.
- Sexton, K., Adgate, J.L., Church, T.R., Greaves, I.A., Ramachandran, G., Fredrickson, A.L., et al., 2003. Recruitment, retention, and compliance results from a probability study of children's environmental health in economically disadvantaged neighborhoods. *Environ. Health Perspect.* 111, 731–736.
- Sexton, K., Adgate, J.L., Church, T.R., Ashley, D.L., Needham, L.L., Ramachandran, G., et al., 2005. Children's exposures to volatile organic compounds as determined by longitudinal measurements in blood. *Environ. Health Perspect.* 113, 342–349.
- Wissow, L., Gittelsohn, A., Szko, M., Starfield, B., Mussman, M., 1988. Poverty, race and hospitalization for childhood asthma. *Am. J. Pub. Health* 78, 777–778.